Modern Concepts of Cardiovascular Disease

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HYPERTENSIVE HEART DISEASE

Advances in diagnosis and the therapeutic management of patients do not always follow as closely as they should the progress in our knowledge of disease. There is a lag—often a period of several years—before facts replace time worn fancies in our conceptions. Such is the case in hypertensive heart disease. To bring diagnosis and treatment abreast of the facts, there must be a more general appreciation that a high percentage (60 to 70%) of individuals with so-called essential hypertension are destined to develop myocardial insufficiency and die of heart failure. About 20% of hypertensive patients succumb to a cerebral accident (hemorrhage or thrombosis) and approximately 10% only die of renal insufficiency. In other words, heart failure is by all odds the most likely contingency in a patient with hypertension.

The hypertensive heart fails for two chief reasons: (1) It carries a much greater load than a normal heart; sooner or later it becomes exhausted, and ultimately fails. (2) The blood supply to the heart wall is often impaired by coronary artery sclerosis. Indeed, the associated coronary disease is frequently the most important factor determining the clinical picture. Thus some hypertensive patients may have anginal seizures or attacks of coronary thrombosis. Others run a progressive down-hill course from congestive failure and at autopsy one finds extreme myocardial fibrosis secondary to more or less severe coronary disease. In many patients, therefore, the capacity of the heart to meet the demands of an elevation in blood pressure—the period of time that the patient is able to carry on—depends chiefly on the state of the coronary arteries. Since this factor is a variable one, one sees at one extreme patients with hypertension able to lead fairly active lives for many years, at the other, individuals who succumb rapidly to heart failure.

The changes found in the hypertensive heart at autopsy are usually simple. The heart is as a rule heavier than normal (250 to 300 grams). Weights about 500 grams are common, although some attain a weight of 750 or even a thousand grams. Most of the increase in weight is due to a thickening of the left ventricle. With the onset of failure, the left ventricle, as well as the other heart chambers, undergoes more or less dilatation. The mitral ring is widened and a relative mitral insufficiency results which is manifested by a blowing systolic murmur at the apex, so commonly heard in failing hypertensive hearts. Contrary to a prevalent conception, there is no actual inflammation—chronic myocarditis—of the heart muscle in uncomplicated cases. Such changes as are found microscopically, patchy areas of fibrosis and slight infiltration with lymphocytes and plasma cells, are secondary to the associated coronary disease. In a word, the pathological findings in the hypertensive heart are those of an organ which has withstood a long siege. It often doubles, and in some instances trebles its normal weight in an attempt to maintain an adequate circulation in the face of great odds. Sooner or later, its reserve is exhausted and we see the appearance of peripheral circulatory failure. Under these circumstances it is not necessary to invoke a chronic inflammatory process in the myocardium to explain the heart's exhaustion. The wonder is, not that the average hypertension heart fails, but that it carries on as long as it does.

So long as the heart is able to meet the demands

of an elevated blood pressure, there are no symptoms of hypertensive heart disease. True, the heart hypertrophied from whatever cause, does not possess a normal reserve, but for all ordinary exertion the patient may be quite unaware of any cardiac insufficiency. The commonest symptom of early failure is dyspnea on exertion. A slight swelling of the ankles toward evening, often ascribed by the patient to age or overweight, is frequent. Another early symptom of left heart failure, particularly in patients who live sedentary lives, is attacks of nocturnal dyspnea, sometimes called cardiac asthma. These attacks vary widely in severity. In mild ones the patient may be awakened by a paroxysmal cough or a sensation of smothering. He may sit up in bed, bring up a little viscid mucus, and fall asleep again. In severe attacks, the dyspnea may be extreme, the breathing noisy, and the face ashen or cyanotic. Paroxysmal coughing may bring up the characteristic pink, frothy sputum of pulmonary edema. The physician must be constantly on the look-out for these earlier manifestations of heart failure because a correct diagnosis and proper therapeutic management at this stage may spare the patient a more serious breakdown.

The therapy of hypertensive heart disease may be considered here under two headings: (1) the treatment of the patient before signs and symptoms of heart failure develop and (2) the management of the case after failure appears. During that period, varying from months to years, in which there are no cardiac symptoms, the informed physician is guided by certain fundamental concepts. For example, he knows that all the while the heart is bearing the brunt of the burden, and that its capacity to withstand the strain will in the majority of cases determine the patient's expectancy. Therefore all therapeutic endeavors are directed toward sparing the heart. Factors known to influence blood pressure, such as mental or emotional strain, obesity, excesses of all kinds, are controlled within reasonable limits. Often the hopeful assurance on the part of the physician, a vacation, a simple sedative insuring a few nights sleep, or even in these times, a rise in the market, may be responsible for an appreciable fall in blood pressure, which is still too often regarded as a specific effect of some medicine. Needless to say that the heart burdened by hypertension must be spared the load of undue physical exertion, so that a curtailment in the patient's activities is indicated. The most reliable guide for this is the amount of exertion that the patient can do without undue breathlessness or fatigue. Living within these limits and controlling the factors mentioned above as influencing blood pressure, the patient is doing all that is known at present to prolong his days. We have as yet no cure for hypertension.

With the development of myocardial insufficiency, we may again be guided by modern concepts in the diagnosis and treatment. Aware of the extra load the heart has been carrying, its exhaustion is anticipated. Therefore, such early manifestations as dyspnea on exertion, mild attacks of cardiac asthma, puffiness about the ankles, should be ascribed to heart failure rather than to bronchitis, asthma, or kidney disease. At this stage, a period of bed-rest is of paramount importance. Sedatives to guarantee sleep, with moderate doses of digitalis and caffeine are useful. If auricular fibrillation is present, the ventricular rate should be brought to normal and maintained with adequate digitalis dosage. Sooner or later many hypertensive patients develop congestive failure and the management of this complication differs in no fundamental way from that of advanced heart failure from other causes. Unfortunately the older concept of the renal origin of hypertension still survives to influence the diagnosis and treatment in such cases, and a failing heart is frequently neglected because dropsy and an albuminous urine are regarded as kidney disease rather than chronic passive congestion from heart failure. Both before and after the stage of heart failure, the patient with hypertension is too often regarded as having, or destined to develop renal insufficiency. How frequently the first advice given the patient concerns a dietary restriction! Protein is thought to be harmful, particularly that in the form of red meat. Eggs are given sparingly but a little white meat and fish are usually permitted. Since the vast majority of hypertensive patients have no inability to utilize protein, over-zealous attempts at protein restriction are not only not indicated, but may do actual harm. There are no reliable clinical or experimental data to prove that the ingestion of a normal amount of protein in food has any influence on blood pressure or on the kidneys in hypertension.

Finally, as mentioned above, coronary arteriosclerosis is frequently associated with hypertension and may therefore at any time dominate the clinical picture with attacks of angina pectoris or coronary thrombosis, but the presence of hypertension is no contraindication to the treatment of these complications as they arise.

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